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EXAMINER	
LONG, SCOTT	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/560,268	KLEINSCHMIDT ET AL.	
	Examiner	Art Unit	
	Scott D. Long	1633	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *30 January 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *11-20* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *11-20* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *3/28/2008*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

The examiner acknowledges receipt of Applicant's Remarks, claim amendments, and specification amendments, filed on 30 January 2008.

Claim Status

Claims 1-10 are cancelled. Claims 1-8 were withdrawn from further consideration by the Examiner, pursuant to 37 CFR 1.142(b), as being drawn to non-elected inventions, there being no allowable generic or linking claim. Claims 11-20 are newly submitted. Claims 11-20 are under current examination.

Information Disclosure Statement

The Information Disclosure Statements (IDS) filed on 28 March 2008 consisting of 2 sheet(s) are in compliance with 37 CFR 1.97. Accordingly, examiner has considered the Information Disclosure Statements.

Priority

This application claims benefit as a 371 of PCT/EP04/006222 (filed 6/9/2004). The application also claims benefit from foreign application EPO 03013169.2 (filed 6/11/2003). The instant application has been granted the benefit date, 11 June 2003, from the application EPO 03013169.2.

Sequence Compliance - Drawings

The disclosure is objected to because of the following:

The specification contains sequence disclosures (**Figure 1**) that are encompassed by the definitions for nucleotide and/or amino acid sequences set forth in 37 CFR 1.82(a)(1) and (a)(2) but are not present in the Sequence Listing and/or identified in the specification by sequence identifier numbers. Applicant must provide sequence identifiers, in the case that these sequence identifier numbers. Applicant must provide sequence identifiers, in the case that these sequences are not included in the original sequence submission, a paper copy and a computer readable copy of the sequence Listing and a statement that the content of the paper and computer readable copies are the same and were applicable, include no new matter, as required by 37 CFR 1.821(e) or 1.821(f) or 1.821(g) or 1.825(b) or 1.825(d). A full response to this Office Action must include complete response to the requirement for a Sequence Listing. **The detailed description of Figure 1, should be amended to include the proper SEQ ID NO for the sequence depicted in Figure 1.** The examiner has noted the amendments to the specification which have incorporated proper reference to various SEQ ID NOs. However, the examiner does not find the new specification amendments sufficient to overcome the objection based on Figure 1.

Appropriate correction is required.

Response to Arguments - Claim Rejections 35 USC § 101

Applicant's arguments (Remarks, page 5) and Claim amendments, filed 30 January 2008, with respect to claims 9-10 have been fully considered and are persuasive. The rejections of Claims 9-10 under 35 USC 101, have been made moot by the cancellation of claims 9-10. Therefore, the examiner hereby withdraws the rejection of claims 9-10 under 35 USC 101.

Response to Arguments - Claim Rejections 35 USC § 112

Response to Arguments – 35 USC 112, second paragraph

Applicant's arguments (Remarks, pages 5-6) and Claim amendments, filed 30 January 2008, with respect to claims 9-10 have been fully considered and are persuasive. The rejections of Claims 9-10 under 35 USC 112, second paragraph, have been made moot by the cancellation of claims 9-10. Therefore, the examiner hereby withdraws the rejection of claims 9-10 under 35 USC 112, second paragraph.

Response to Arguments – Written Description (35 USC 112, first paragraph)

Applicant's arguments (Remarks, page 6) and Claim amendments, filed 30 January 2008, with respect to claims 9-10 have been fully considered and are persuasive. The rejections of Claims 9-10 under 35 USC 112, first paragraph (written description), have been made moot by the cancellation of claims 9-10. Therefore, the examiner hereby withdraws the rejection of claims 9-10 under 35 USC 112, first paragraph (written description).

Response to Arguments - Claim Rejections 35 USC § 103

Applicant's arguments (Remarks, page 6-7) and Claim amendments, filed 30 January 2008, with respect to claims 9-10 have been fully considered and are persuasive. The rejections of Claims 9-10 under 35 USC 103(a) as obvious over Bartlett et al. (US Patent 6,962,815, issued 8 November 2005) in view of Kaplitt et al. (US Patent, 6,162,796, issued 19 December 2000), have been made moot by the cancellation of claims 9-10. Therefore, the examiner hereby withdraws the rejection of claims 9-10 under 35 USC 103.

NEW GROUNDS OF REJECTION

Claim Rejections - 35 USC § 112

Claim 11 is rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. The instant specification teaches "Mutational analysis of AAV-2 capsid proteins VP1, VP2 and VP3, respectively showed that a group of basic amino acids (arginines 484, 487, 585, 588 and lysine 532; numbering according to the numbering based on W-1 protein id AAC03780.1 NCBI accession No. AF043303) contributes to heparin and HeLa cell binding." (page 4, lines 8-12). Since instant claim 11 does not indicate which strain of AAV is being used to determine the mutation sites, a skilled artisan would not necessarily know whether any AAV other than AAV-2 could

be used in this invention. This is particularly important because the capsid homology of serotypes AAV4 and AAV5 are more distantly related to capsids of serotypes AAV1, AAV2, and AAV3. Furthermore, the primary receptor for AAV2 is heparan sulfate proteoglycan, whereas the receptors for AAV4 and AAV5 are likely different (Xie et al. PNAS 2002; 99(16):10405-10410).

Additionally, the specification teaches, "According to the recently resolved atomic structure for AAV-2, arginines 484, 487 and lysine 532 on one site and arginines 585 and 588 on the other site belong to different capsid protein subunits." (page 4, lines 14-17). Since the specification teaches that the R484 mutation and R585 mutations are on different capsid protein subunits, the examiner concludes that instant claim 1 is not particularly pointed out and distinctly claimed. It seems that this limitation that the mutations must be on different capsid proteins in an important feature of the invention. Clarification of the claim is requested.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 11-20 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of gene delivery of a double-mutant (R484E/R585E) adeno-associated virus to heart tissue, does not reasonably provide enablement for gene therapy (i.e., expression of a therapeutic gene in the target tissue

such that a effective dose induces biological effect associated with improved health) for any tissue. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The factors to be considered in determining whether undue experimentation is required are summarized *In re Wands* 858 F.2d 731, 8 USPQ2nd 1400 (Fed. Cir, 1988). The Court in *Wands* states: "Enablement is not precluded by the necessity for some 'experimentation.'" Clearly, enablement of a claimed invention cannot be predicated on the basis of quantity of experimentation required to make or use the invention. "Whether undue experimentation is needed is not a single simple factual determination, but rather is a conclusion reached by weighing many factual considerations." (*Wands*, 8 USPQ2d 1404). The factors to be considered in determining whether undue experimentation is required include: (1) the quantity of experimentation necessary, (2) the amount or direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims. While all of these factors are considered, a sufficient amount for a prima facie case is discussed below.

SCOPE OF THE INVENTION

The breadth of the claims encompasses a genus of therapies for any non-hepatic tissue. As discussed supra, the specification fails to describe the genus of "therapies"

(i.e., which specific diseases are being treated and with which genes) and also fails to describe a genus of gene delivery methods for non-hepatic tissues other than for heart tissue. Therefore, undue experimentation would be required to discover these methods. Additionally, instant claim 1 implies that the reduced or eliminated heparin binding function of the mutant AAV particle may be responsible for some therapeutic effect. Specific diseases associated with this lack of heparin binding function is not described in the specification. The specification only discloses and provides guidance for methods of gene delivery to cardiac tissue by a mutant AAV particle comprising R484E and R585E mutations.

GUIDANCE & WORKING EXAMPLES

The specification does not provide guidance for or a working example for any gene therapy. Rather, the specification teaches cardiac tissue-specific targeting of a mutant AAV comprising R484E and R585E mutations. Example 6 teaches, recombinant AAV-2 mutant #17 (comprising R484E and R585E mutations) was delivered by tail vein injection and the only tissue where reporter gene expression was detected only in cardiac tissue, while wild-type AAV-2 administered by the same method showed reporter gene function to both cardiac tissue and liver tissue (page 21). Example 6 also shows that neither wild type or mutant AAV demonstrated sufficient targeting to permit detectable levels of reporter in other tissues analyzed. A simple conclusion of this example, is that the R484E/R585E mutant AAV can target cardiac tissue and is unable to target hepatic tissue. There are no other working examples in

the specification with show a "therapeutic" level of any protein expressed after IV delivery of the mutant AAV. Additionally, based on the teachings of the specification, the R484E/R585E mutant AAV does not seem to be capable of targeting non-cardiac tissue. The absence of working examples directed to expression of therapeutic levels of any protein necessitates further experimentation.

STATE OF THE ART & QUANTITY OF EXPERIMENTATION

The nature of the invention being gene therapy, the state of the prior art is not well developed and is highly unpredictable. Verma et al (Nat. 1997 Sep; 389:239-242) states that out of the more than 200 clinical trials currently underway, no single outcome can be pointed to as a success story (page 239, col. 1). For instance, numerous factors complicate the gene therapy art which have not been shown to be overcome by routine experimentation. Eck et al. (Phar Basis Ther 1995; 77-101) explains, the fate of the DNA vector itself (volume of distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used, the protein being produced, and the disease being treated. (paragraph bridging pages 81-82) Verma et al. states

that one major obstacle to success has been the inability to deliver genes efficiently and obtain sustained expression (see Verma et al., page 239, col. 3).

CONCLUSION

In conclusion, based upon the nature of the claimed invention, the state of the art, the unpredictability found in the art, the teaching and working examples provided, and the breadth of the claims that it would require undue experimentation to practice the invention beyond the scope of a method of gene delivery of a double-mutant (R484E/R585E) adeno-associated virus to heart tissue.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

Claims 11-20 are rejected under 35 U.S.C. 102(e) as being anticipated by Warrington et al. (US2006/0088936, published 27 April 2006).

Claim 11 is directed to a method of gene therapy in a non-hepatic tissue of a patient, comprising delivering to a patient an AAV vector or an AAV particle having a

capsid encoded by the AAV vector, wherein the AAV vector carries at least one mutation in a heparin-binding motif of a capsid protein and causes a reduced or eliminated heparin binding function, wherein said mutation is an amino acid substitution at amino acid position arginine 484 and/or arginine 585. Warrington et al. teach "recombinant adeno-associated viral (rAAV) vectors having mutations in one or more capsid proteins. Exemplary vectors are provided that have altered affinity for heparin or heparin sulfate" (abstract). Warrington et al. teach rAAV vectors…comprising…R585A …mutation, affinity for heparin sulfate binding by the vector was eliminated." (page 3, parag.0028) Warrington et al. rAAV comprising identify mutant R484A as having reduced heparin binding (page 8, parag.0075). Warrington et al. teach delivery of AAV vectors by intravenous administration.

Claims 12-14 are directed to specific amino acid mutations of the capsid. Warrington et al. teach conservative amino acid substitutions (claim 12), including R484A (claim 13-14).

Claims 15-18 are directed to further limitations of the claims, wherein the AAV vector is AAV-2 (claim 15); capsid proteins are VP1 (claim 17); VP1, VP2, or VP3 (claim 16); number of amino acid position is according to VP1 (claim 18). Claim 20 is directed to system delivery of the AAV. All of the limitations of claims 15-20 are taught by the cited references.

Claim 19 is directed to the method of claim 11, wherein said non-hepatic tissue is a hear muscle tissue. Although, Warrington et al. does not recognize that their vectors will target specifically to cardiac tissue, they do teach that these mutations will alter

tropism of the AAV. Therefore, the examiner believes the limitations of claim 19 are inherent in the structure of the AAV mutants taught by Warrington.

Accordingly, Warrington et al. anticipated the instant claims.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 11 and 15-20 are rejected under 35 U.S.C. 103(a) as being unpatentable over Bartlett et al. (US Patent 6,962,815, issued 8 November 2005) in view of Kaplitt et

al. (US Patent, 6,162,796, issued 19 December 2000) and further in view of Wu Xiao (PhD Dissertation 2002, University of Florida).

Claim 11 is directed to a method of gene therapy in a non-hepatic tissue of a patient, comprising delivering to a patient an AAV vector or an AAV particle having a capsid encoded by the AAV vector, wherein the AAV vector carries at least one mutation in a heparin-binding motif of a capsid protein and causes a reduced or eliminated heparin binding function, wherein said mutation is an amino acid substitution at amino acid position arginine 484 and/or arginine 585.

Bartlett et al. teach, "Recent research on AAV has therefore involved attempts to modify the viral genome. As the range of cells that AAV will infect is so broad, some researches have focused on modifying the virus so that it targets specific types of cells for infection. The cellular range or tropism of the virus is determined by the binding of AAV capsid protein(s) to receptor and/or coreceptor proteins expressed on the surface of target cells. Heparin-sulfate proteoglycans (HSPG) is the primary cellular attachment receptor for AAV2." (col.2, lines 11-19). Bartlett et al. further teach, "AAV vectors of the invention that exhibit an altered cellular tropism may differ from wild type in that the natural tropism of AAV may be reduced or abolished" (col.4, lines 41-65). The instant application states, "Mutational analysis of AAV-2 capsid proteins VP1, VP2 and VP3, respectively showed that a group of basic amino acids (arginines 484, 487, 585, 588 and lysine 532; numbering according to the numbering based on VP1 protein id AAC03780.1 NCBI accession No. AF043303) contributes to heparin and HeLa cell binding. These amino acids are positioned in three clusters on the threefold spikes of

the AAV-2 capsid." (page 4, 1st parag.). Bartlett et al. further teach amino acids 584 and 588 of VP1 as being important to heparin binding (col.17, lines 1-7 and col.41, line 26). This AAV vector contains at least one mutation to the capsid proteins in amino acid positions 470 to 592, which affects heparin binding. Bartlett et al. teach "The AAV-RGD vectors A588-RGD4C-eGFP and A588-RGD4CGLS were tested for their ability to target gene transfer to the ovarian cell lines as described in Example 9...were able to more efficiently direct gene transfer...compared to wild type AAV vector containing unmodified capsid" (col.19, lines 56-64).

Bartlett et al. do not teach specific delivery of AAV to heart muscle tissue and do not explicitly teach AAV mutants comprising amino acid substitutions at amino acid position arginine 484 and/or arginine 585.

Kaplitt et al. teach, "AAV naturally infects heart muscle...AAV vectors can yield long-term expression not observed with other systems" (parag.0025) and "the present invention results in gene transfer and expression to a wide area of heart muscle" (parag.0027).

Kaplitt et al. do not teach the specific mutations of capsid proteins and its corresponding effect on heparin-sulfate binding proteins as required by the instant claims.

However, Wu Xiao et al. teach "to increase the targeting of rAAV vectors...1) reducing the natural tropism of AAV, and 2) increasing the tissue specificity of AAV...[have been attempted by] groups [of researchers who] have been trying to locate sites of AAV capsid for receptor binding and the sites exposed on the surface of the

capsid by doing extensive capsid mutagenesis experiments" (page 14, lines 6-10). Wu Xiao also teaches “double mutants at amino acid 585 and 588 of AAV capsid protein abolish its heparin binding activity” (page 14, lines 19-20).

Claims 15-18 are directed to further limitations of the claims, wherein the AAV vector is AAV-2 (claim 15); capsid proteins are VP1 (claim 17); VP1, VP2, or VP3 (claim 16); number of amino acid position is according to VP1 (claim 18). Claim 19 is directed to the use according to claim 11, wherein said non-hepatic tissue is heart muscle tissue. Claim 20 is directed to system delivery of the AAV. All of the limitations of claims 15-20 are taught by the cited references.

It would have been obvious to one skilled in the art to use an AAV vector having at least one mutation to the capsid proteins in amino acid positions 484 and/or 585, which affects heparin binding in a method of gene therapy to heart muscle tissue.

Regarding the rationale for combining prior art elements according to known methods to yield predictable results, all of the claimed elements were known in the prior art and one skilled in the art could have combined the element as claimed by known methods with no change in their respective functions, and the combination would have yielded predictable results to one of ordinary skill in the art at the time of the invention. Each of the elements (mutations to AAV-2 capsid protein at position 585 and AAV having a specificity for heart muscle tissue) are taught by Bartlett, Kaplitt, and Wu Xiao. Wu Xiao, in particular, teaches that mutations of capsid proteins are capable of limiting the range of cellular targeting by AAV.

Therefore the methods as taught by Bartlett et al. in view of Kaplitt et al. and further in view of Wu Xiao would have been *prima facie* obvious over the method of the instant application.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

No claims are allowed.

Examiner Contact Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to **Scott Long** whose telephone number is **571-272-9048**. The examiner can normally be reached on Monday - Friday, 9am - 5pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, **Joseph Woitach** can be reached on **571-272-0739**. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/SDL/ Scott Long Patent Examiner, Art Unit 1633	/Janet L. Epps-Ford/ Primary Examiner, Art Unit 1633